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A PART OF A DISSERTATION SUBMITTED TO THE FACULTY OF THE DIVISION OF BIOLOGICAL SCIENCES IN CANDIDACY FOR THE DEGREE OF DOCTOR OF PHILOSOPHY.

Department of Physiology

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Acute Effects Resulting from Downward Traction on the Liver.*

Walter M. Booker, Ph.D., Prairie View, Texas

IT IS BECOMING increasingly well-known that manipulation or distention of the abdominal viscera may cause a pronounced fall in blood pressure, and this attended in many instances by a temporary inhibition of respiration, lasting from 15 to 30 seconds. In a series of experiments Beard and Blalock[2] in 1931, Johnson and Blalock[7] in the same year, and Blalock[3] in 1933 studied the cause and treatment of shock produced by manipulation of the intestine. Since that time a number of investigators have reported confirmatory results of the work of Blalock and his co-workers.

Introduction

IN 1932 Luckhardt, Alpert and Smith[8] showed that pulling on the liver caused a sharp fall in blood pressure and an inhibition of respiration. And in 1941 Booker[4] reported in outline form and without interpretation that when downward traction on the liver was maintained for long periods of time (2½ to 3½ hours) animals were thrown into circulatory failure and death resulted. The importance and practicality of this work may be seen when it is recalled how the liver is often "rotated" in removal of the gall-bladder or drainage of cysts. The irregular, shallow and spasmodic respiratory movements are disturbing to both the surgeon and the anesthetist. Also the effects of such manipulations simulate the findings of a completely decompensated heart, with passive congestion in all parts of the body below the diaphragm. Furthermore, the whole problem of shock or circulatory failure has become particularly of utmost importance in the last few years; and it is felt that such experiments as these offer a simple and convenient way of studying the problem of circulatory failure.

Methods of Procedure

THIS GROUP of experiments involved the use of more than forty dogs. The animals were anesthetized usually with nembutal (in only a few cases was barbital used) and the carotid artery was connected by a cannula to a mercury manometer. Respiration was recorded by the usual pneumograph-tambour set-up. An incision was made at the tip of the sternum and extended along the midline to the lower region of the abdomen. For long periods of traction a brass "hand" was placed over the liver and was tied by a string to the opposite end of the operating table, so that a constant tension could be maintained. In order to prevent loss of heat the abdomen was covered with towels; and in some instances a lamp was placed just above the abdomen throughout the period

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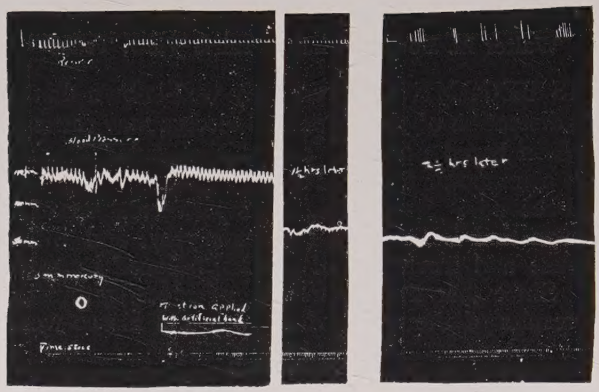

Fig. 1. A typical experiment showing circulatory failure from continuous traction on the liver. Blood pressure intervals: 50 mm. Time: 5 secs.

of traction. Blood cell counts were made in the usual way; and hematocrit values were obtained by use of the Van Allen hematocrit tubes.

Results and Interpretations

OBSERVATION OF *Fig.* 1 will reveal the typical picture which is produced when traction on the liver is maintained continuously for 2½ to 3½ hours. In some cases death may occur sooner or later than the above time. It will be noted that the arterial blood pressure presents three definite phases, which the author has seen fit to term conveniently (1) period of acute drop; (2) period of attempted compensation, and (3) period of slow decline and complete failure of circulation. The first period is the result of mechanical obstruction of the vena cava and the hepatic vein, reducing the venous return to the heart and consequently causing a reduction in the cardiac output. With the chest open (artificial respiration) when traction is exerted on the liver, direct observation of the vena cava shows a marked reduction in the cross-sectional area of this vessel. Furthermore, Herrick, Mann, Essex and Baldes[6] in 1938 visualized the intrathoracic vena cava in dogs which had been treated with thorotrast, and noted when traction was exerted on the stomach or

TABLE I

A typical experiment showing changes in blood pressure (arterial), hematocrit values and blood counts during continuous traction on the liver.

Period	Blood Pressure	Hematocrits		Cell Counts
		Cells	Plasma	
Before Traction	160	40	60	5,500,000
30 min. Traction	55	50	50	6,400,000
1 hour Traction	55	55	45	6,800,000
1½ hours Traction	65	60	40	8,800,000
2 hours Traction	50	65	35	9,600,000
2½ hours	30	55	45	7,280,000

on the liver there was "marked reduction of the diameter of the vena cava." The period of compensation is contributed to by a number of factors: (1) carotid sinus reflex, (2) vasoconstriction from increased venous pressure (McDowell's reflex[9 11]), and (3) contraction of the spleen with output of blood into the general circulation. The third period, that of slow decline and complete circulatory failure, is the point of greatest concern and speculation experimentally and clinically. This author's hypothesis is that traction on the liver causes a rise in venous pressure (*Fig.* 2) in the abdominal veins, which results in extravasation of fluid from blood vessels, causing hemoconcentration and consequent decrease in blood volume. That hemoconcentration occurs is indicated

Fig. 2. A typical experiment showing increased venous pressure and decreased arterial pressure when traction is exerted on the liver.

by a rise in hematocrit values and in red cell count (*Table* 1). Although no blood volume studies as such were made, all animals showed large quantities of hemorrhagic fluid in the abdominal cavity after an hour or more of traction. This, it seems to the author, would be indirect evidence, at least, of decrease in blood volume. This is, of course, contributed to by venous stasis and probable resultant anoxia at the capillaries. A close observation of *Table* 1 shows that late in the period of traction the hematocrit values and the cell count decrease. Katz and his co-workers[10], producing shock in dogs by ligation of the iliac vein, have made a similar observation; and they attribute the fall in the hematocrit values to a pressure gradient at the tissues, causing movement of the extravasated fluid back to blood. Apparently, however, the fluid does not move back in sufficient quantity to cause a rise in blood volume to the point of facilitating a rise in arterial blood pressure. So that in a course of time, the arterial pressure is seen at low levels (40 to 60 mm.

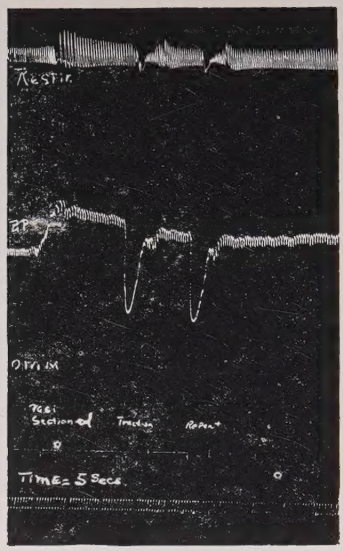

Fig. 3. A typical experiment showing inhibition of respiration upon traction on the liver, vagi sectioned.

and lower); the pulse is rapid and thready; there is respiratory failure and death soon follows. This is a type of circulatory failure described by Harrison[5] as forward circulatory failure.

Interesting, but of no serious consequence to the animal, is the temporary inhibition of respiration. Luckhardt, Alpert and Smith, aforementioned, offered the explanation that in part this inhibition was reflex

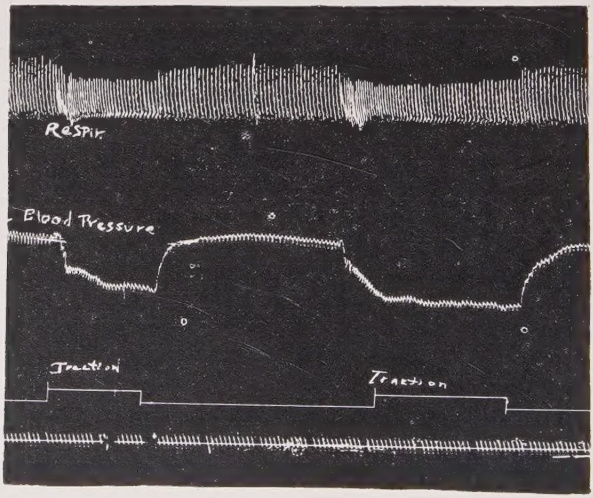

Fig. 4. An experiment showing the absence of the respiratory inhibition when traction was exerted. The liver had been previously infiltrated with procaine-epinephrine solution (2%). Vagi sectioned.

in nature over the vagus nerve (Hering-Breuer reflex), as the period of inhibition could be reduced, but not removed, after double vagotomy. And Bamberger[1] described a similar inhibition of respiration when the vena cava, the hepatic or the hepatic portal veins were pulled. While double vagotomy plus cocainization of the veins removed the inhibition completely, double vagotomy alone failed to do so. It can be seen in *Fig.* 3 that traction on the liver, vagi sectioned, gives a shorter inhibition of respiration than when the vagi are intact (compare with *Fig.* 1). When, however, the liver was infiltrated (at random over the large lobes) with 2 per cent procaine-epinephrine solutions, vagi cut, no inhibition of respiration occurred upon pulling on the liver (*Fig.* 4).

The explanation seems warranted that in addition to the vagal pathway, inhibitory impulses are carried over sympathetic fibers, the endings of which are blocked if procaine is injected. It is further significant that

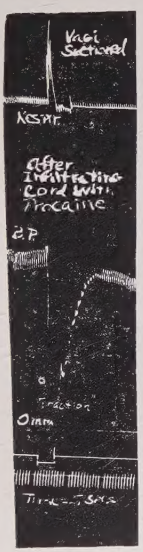

Fig. 5. A typical experiment showing traction on the liver following infiltration of the cord (at splanchnic origin), vagi sectioned. Note there is no inhibition of respiration.

procainization of the spinal cord, vagi cut, also removes the inhibition when traction is exerted (*Fig.* 5).

Summary and Conclusions

FIRST. Experiments have been described showing the acute effects resulting from downward traction on the liver. (2) Evidence was presented to show that continuous traction on the liver for 2½ to 3½ hours results in circulatory failure and death of the animals. Such animals before death show a marked hemoconcentration, hemorrhagic fluid in the abdominal cavity, a low arterial and a high venous pressure, with a rapid and thready pulse. (3) The increased venous pressure (in abdominal region) causes extravasation of fluid from blood, reducing blood volume. (4) The evidence seems to show that the temporary

respiratory inhibition is reflex over both the vagi and the sympathetics, as the duration of the inhibition may be reduced by sectioning the vagi. If, however, the vagi are sectioned and the sympathetics are blocked the inhibition does not occur. (5) Liver traction experiments are suitable and worthwhile to study the ramifying effects of circulatory failure.

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